REFERENCES

1. Bergsagel DE: Macroglobulinemia, *In* Williams JW, Beutler E, Erslev AJ, et al (Eds): Hematology, 3rd Ed. New York, McGraw-Hill, 1983, pp 1104-1108
2. McCallister BD, Bayrd ED, Harrison EG, et al: Primary macroglobulinemia: Review with report of 31 cases. Am J Med 1967; 43:394-434
3. MacKenzie MR, Fudenberg HH: Macroglobulinemia: An analysis of 40 patients. Blood 1972; 39:874-889
4. Carter P, Koval JJ, Hobbs JR: The relation of clinical and laboratory findings to the survival of patients with macroglobulinemia. Clin Exp Immunol 1977; 28:241-249
5. Stein RS, Ellman L, Bloch KJ: The clinical correlates of IgM M-components: An analysis of 34 patients. Am J Med Sci 1975; 269:209-216
6. Krajny M, Pruzanski W: Waldenström's macroglobulinemia: Review of 45 cases. Can Med Assoc J 1976; 114:899-905
7. Perkins HA, MacKenzie MR, Fudenberg HH: Hemostatic defects in dysproteinemias. Blood 1970; 35:695-707
8. MacKenzie MR, Brown E, Fudenberg HH, et al: Waldenström's macroglobulinemia: Correlation between expanded plasma volume and increased serum viscosity. Blood 1970; 35:394-408
9. McGrath MA, Penny R: Paraproteinemia: Blood hyperviscosity and clinical manifestations. J Clin Invest 1976; 58:1155-1162
10. Morel-Maroger L, Basch A, Danon F, et al: Pathology of the kidney in Waldenström's macroglobulinemia: Study of 16 cases. N Engl J Med 1970; 283:123-129
11. Martello OJ, Schultz DR, Pardo V, et al: Immunologically-mediated renal disease in Waldenström's macroglobulinemia. Am J Med 1975; 58:567-575
12. Rausch PG, Herion JC: Pulmonary manifestations of Waldenström's macroglobulinemia. Am J Hematol 1980; 9:201-209
13. Dellagi K, Dupouey P, Brouet JC, et al: Waldenström's macroglobulinemia and peripheral neuropathy: A clinical and immunologic study of 25 patients. Blood 1983; 62:280-285
14. Case DC: Combination chemotherapy (M-2 protocol) for Waldenström's macroglobulinemia: Preliminary report. Blood 1982; 59:934-937

Acute Aortic Defects

MANY PATIENTS who are now being seen in the emergency room with chest pain are being considered for thrombolytic therapy. Obviously, the administration of thrombolytic therapy, whether it is streptokinase or tissue plasminogen activator in a patient with aortic dissection masquerading as a myocardial infarction, has very important implications in terms of patient welfare, as well as medicolegal implications. I suspect we may well see more and more of these reports, since the message that we are receiving from people very active in this area is that if we are going to make a significant impact and salvage myocardium during the acute phase of myocardial infarction, the thrombolytic therapy has to be given very early, and generally, it is given without catheterization and confirmation of coronary occlusion.

Now, the relationship of the site of pain to the origin of dissection is an important point. Most proximal dissections will be associated with anterior chest pain. In contrast, patients with distal dissections, type III dissections versus type I or type II, typically will present with back pain—posterior pain. Again, though, there is some variability, and the key is to ask the patient to describe the quality of pain: it is usually excruciating; it is usually tearing; it is usually maximal in intensity at outset.

Other less common presenting symptoms include syncope. In patients with dissection who present with syncope, the mechanism is almost always acute pericardial tamponade, although, rarely, significant hemorrhage into either the pleural space or the mediastinum will cause syncope. But syncope means pericardial tamponade. The presence of a pericardial effusion in a patient with a high probability of dissection is associated with an extremely high mortality. Roughly 75% of patients with pericardial effusions and dissections are dead within 24 hours if surgical therapy is not carried out.

Transient ischemic attack (TIA) and stroke, because of branch vessel occlusion; pulse loss, with or without ischemic pain; and also, congestive heart failure—these patients almost always have aortic regurgitation.

Finally, painless dissection can occur. It is very rare, and the majority of these patients at the time of presentation exhibit disturbed consciousness. If they were able to give a history, whether or not they would complain of chest pain is not clear in reviewing the history.

—ROBERT S. GIBSON, MD

Extracted from *Audio-Digest Surgery*, Vol. 34, No. 19, in the Audio-Digest Foundation's series of tape-recorded programs. For subscription information: 1577 E Chevy Chase Dr, Glendale, CA 91206